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PATENT

STIFFENING PHARYNGEAL WALL TREATMENT

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CROSS-REFERENCE TO RELATED APPLICATION

This application is a continuation of application Serial No. 10/237,149 filed September 6, 2002, which is a continuation-in-part of application Serial No. 10/066,967 filed February 4, 2002; which applications are incorporated herein by reference.

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BACKGROUND

1. Field of the Invention

This invention is directed to methods and apparatuses for treating the pharyngeal wall of a patient. More particularly, this invention pertains to method and apparatus for treating a pharyngeal wall area as part of a sleep apnea treatment.

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2. Description of the Prior Art

Sleep apnea and snoring are complex phenomena. Commonly assigned U.S. Pat. No. 6,250,307 describes various prior techniques and discloses a novel treatment for such conditions (including a permanent palatal implant).

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These prior art teachings include Huang, et al., "Biomechanics of Snoring", Endeavour, p. 96 – 100, Vol. 19, No. 3 (1995). That publication estimates that up to 20% of the adult population snores habitually. Snoring can be a serious cause of marital discord. In addition, snoring can present a serious health risk to the snorer. In 10% of habitual snorers, collapse of the airway during sleep can lead to obstructive sleep apnea 25 syndrome. Id. In addition to describing a model for palatal flutter, that publication also describes a model for collapse of the pharyngeal wall.

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Notwithstanding efforts have been made to treat snoring and sleep apnea. These include palatal treatments such as electrical stimulation of the soft palate. See, e.g., Schwartz, et al., "Effects of electrical stimulation to the soft palate on snoring and obstructive sleep apnea", J. Prosthetic Dentistry, pp. 273 – 281 (1996). Devices to apply such stimulation are described in U.S. Pat. Nos. 5,284,161 and 5,792,067. Such

devices are appliances requiring patient adherence to a regimen of use as well as subjecting the patient to discomfort during sleep. Electrical stimulation to treat sleep apnea is discussed in Wiltfang, et al., "First results on daytime submandibular electrostimulation of suprathyroidal muscles to prevent night-time hypopharyngeal collapse in obstructive sleep apnea syndrome", International Journal of Oral & Maxillofacial Surgery, pp. 21 – 25 (1999).

Surgical treatments for the soft palate have also been employed. One such treatment is uvulopalatopharyngoplasty (UPPP) where about 2 cm of the trailing edge of the soft palate is removed to reduce the soft palate's ability to flutter between the tongue and the pharyngeal wall of the throat. See, Huang, et al., supra at 99 and Harries, et al., "The Surgical treatment of snoring", Journal of Laryngology and Otology, pp. 1105 – 1106 (1996) which describes removal of up to 1.5 cm of the soft palate. Assessment of snoring treatment is discussed in Cole, et al., "Snoring: A review and a Reassessment", Journal of Otolaryngology, pp. 303 – 306 (1995). Huang, et al., propose an alternative to UPPP which proposal includes using a surgical laser to create scar tissue on the surface of the soft palate. The scar is to reduce flexibility of the soft palate to reduce palatal flutter. RF ablation (so-called Somnoplasty as advocated by Somnus Technologies) is also suggested to treat the soft palate. RF ablation has also been suggested for ablation of the tongue base.

In pharyngeal snoring and sleep apnea, the pharyngeal airway collapses in an area between the soft palate and the larynx. One technique for treating airway collapse is continuous positive airway pressure (CPAP). In CPAP air is passed under pressure to maintain a patent airway. However, such equipment is bulky, expensive and generally restricted to patients with obstructive sleep apnea severe enough to threaten general health. Huang, et al. at p. 97.

Treatments of the pharyngeal wall include electrical stimulation is suggested in U.S. Pat. No. 6,240,316 to Richmond et al. issued May 29, 2001, U.S. Pat. No. 4,830,008 to Meer issued May 16, 1989, U.S. Pat. No. 5,158,080 to Kallok issued October 27, 1992, U.S. Pat. No. 5,591,216 to Testerman et al. issued January 7, 1997 and PCT International Publication No. WO 01/23039 published April 5, 2001 (on PCT International Application No. PCT/US00/26616 filed September 28, 2000 with priority

to U.S. Ser. No. 09/409,018 filed September 29, 1999). U.S. Pat. No. 5,979,456 to Magovern dated November 9, 1999 teaches an apparatus for modifying the shape of a pharynx. These teachings include a shape-memory structure having an activated shape and a quiescent shape. Dreher et al., "Influence of nasal obstruction on sleep-associated breathing disorders", *So. Laryngo-Rhino-Otologie*, pp. 313 – 317 (June 1999), suggests using nasal stents to treat sleep associated breathing disorders involving nasal obstruction. Upper airway dilating drug treatment is suggested in Aboubakr, et al., "Long-term facilitation in obstructive sleep apnea patients during NREM sleep", *J. Applied Physiology*, pp. 2751 – 2757 (December 2001).

10 Surgical treatments for sleep apnea are described in Sher et al., "The Efficacy of Surgical Modifications of the Upper Airway in Adults with Obstructive Sleep Apnea Syndrome", *Sleep*, Vol. 19, No. 2, pp. 156 – 177 (1996). Anatomical evaluation of patients with sleep apnea or other sleep disordered breathing are described in Schwab, et al., "Upper Airway and Soft Tissue Anatomy in Normal Subjects and Patients with

15 Sleep-Disordered Breathing", *Am. J. Respir. Crit. Care Med.*, Vol. 152, pp. 1673 – 1689 (1995) ("Schwab I") and Schwab et al., "Dynamic Upper Airway Imaging During Awake Respiration in Normal Subjects and Patients with Sleep Disordered Breathing", *Am. Rev. Respir. Dis.*, Vol. 148, pp. 1385 – 1400 (1993) ("Schwab II"). In Schwab I, it is noted that apneic patients have a smaller airway size and width and a thicker lateral

20 pharyngeal wall. For reviews of pharyngeal wall thickness and other structure and obstructive sleep apnea, see, also, Wheatley, et al., "Mechanical Properties of the Upper Airway", *Current Opinion in Pulmonary Medicine*, pp. 363 – 369 (November 1998); Schwartz et al., "Pharyngeal airway obstruction in obstructive sleep apnea: pathophysiology and clinical implication", *Otolaryngologic Clinics of N. Amer.*, pp. 911 – 918 (December 1998); Collard, et al., "Why should we enlarge the pharynx in obstructive sleep apnea?", *Sleep*, (9 Suppl.) pp. S85 – S87 (November 1996); Winter, et al., "Enlargement of the lateral pharyngeal fat pad space in pigs increases upper airway resistance", *J. Applied Physiology*, pp. 726 – 731 (September 1995); and Stauffer, et al., "Pharyngeal Size and Resistance in Obstructive Sleep Apnea", *Amer. Review of*

25 *Respiratory Disease*, pp. 623 – 627 (September 1987).

There are contrasting opinions in the medical literature on the mechanisms of OSA. OSA patients are a heterogeneous group; there are differing locations and patterns of pharyngeal collapse for each person. In addition to the physical findings and properties which characterize the pharynx in patients with OSA such as increased

5 collapsibility, increased compliance, increased resistance, and decreased cross-sectional area, the physical properties and spatial relationships of the pharyngeal airway, head, and neck, as well as the neuromuscular integrity of the airway (reflexes affecting upper airway caliber) and mechanisms of breathing control (pharyngeal inspiratory muscle function) must also be considered relevant in their contribution to the mechanism and

10 precipitation of upper airway collapse. Hudgel DW, *Mechanisms of Obstructive Sleep Apnea*. Chest 1992; 101:541-49. Fairbanks DNF, Fujita S, Snoring and Obstructive Sleep Apnea. Raven Press Ltd., New York, 1994.

In general, obstructive apnea occurs during sleep, when the pharyngeal dilator muscle activity (genioglossus, tensor palatini, geniohyoid, stylohyoid) that normally

15 maintains airway patency during inspiration through dilation of the airway, is diminished. (Fairbanks DNF, Fujita S, Snoring and Obstructive Sleep Apnea. Raven Press Ltd., New York, 1994.2, p. 85). When the intraluminal negative pressure of the airway reaches a critical point, the combination of redundant tissues and the loss of pharyngeal muscle tone causes airway collapse during inspiration. Please note,

20 obstruction has been shown to occur during expiration and inspiration (Schwab RJ et al., *Dynamic imaging of the upper airway during respiration in normal subjects*. J Appl Physiol 1993; 74(4):1504-1514. Schwab RJ, *Functional Properties of the Pharyngeal Airway*. Sleep 1996; 19(10):S170-S174. 8, 9); details on how upper airway area changes during the respiratory cycle can be found in the cited literature. Surgical

25 treatments are aimed at eliminating any collapsible tissue in the airway and reducing airway resistance without creating functional impairment of the upper airway structures.

SUMMARY OF THE INVENTION

According to one aspect of the present invention, methods and apparatuses are

30 disclosed for treating a pharyngeal airway having a pharyngeal wall of a patient at least partially surrounding and defining said airway. The method includes selecting an

implant dimensioned so as to be implanted beneath a mucosal layer of the pharyngeal wall and extending transverse to said wall. The implant has mechanical characteristics for the implant, at least in combination with a fibrotic tissue response induced by the implant, to stiffen aid pharyngeal wall to resist radial collapse. The implant is
5 implanted into the pharyngeal wall transverse to a longitudinal axis of the airway.

BRIEF DESCRIPTION OF THE DRAWINGS

- Fig. 1 shows, in cross-section, a naso-pharyngeal area of an untreated patient;
Fig. 2 is the view of Fig. 1 with the soft palate containing an implant in the form
10 of a bolus of micro-beads deposited in a linear path;
Fig. 3 is a frontal view of the patient of Fig. 3 showing an alternative embodiment with micro-beads deposited as spherical deposits;
Fig. 4 is a schematic representation showing a patch for delivering a bolus of micro-beads through a plurality of needles;
15 Fig. 5 is a schematic cross-sectional view (taken generally along line 5 – 5 in Fig. 1) of a pharyngeal airway at a position in a person with the airway defined by opposing portions of a pharyngeal wall and a base of a tongue;
Fig. 6 is a view similar to that of Fig. 1 (only showing patient's right side) with a device according to the present invention placed in the pharyngeal wall and with the
20 pharyngeal wall and the device shown in perspective for ease of illustration;
Fig. 7 is a cross-sectional view taken along section line 7-7 of Fig. 6;
Fig. 8 is a view similar to that of Fig. 6 showing multiple devices placed in the pharyngeal wall;
Fig. 9 is a view similar to that of Fig. 6 showing multiple devices placed in the
25 pharyngeal wall and oriented parallel relative to a longitudinal axis of the air passage;
Fig. 10 is a cross-sectional view taken along section line 10-10 of Fig. 9; and
Fig. 11 is a view similar to that of Fig. 6 with a sheet placed in the pharyngeal wall.

30 DESCRIPTION OF THE PREFERRED EMBODIMENT

A. Physiology Background

Referring now to the several drawing figures, in which identical elements are numbered identically throughout, a description of a preferred embodiment of the present invention will now be provided.

The disclosures of U. S. Pat. No. 6,250,307 and PCT International Publications

- 5 No. WO 01/19301 (PCT/US00/40830) and WO 02/13738 (PCT/US01/24255) are incorporated herein by reference.

Fig. 1 shows, in cross-section, a naso-pharyngeal area of an untreated patient. Fig. 1 shows the nose N, mouth M and throat TH. The tongue T is shown in an oral cavity OC of the mouth. A hard palate HP (containing a bone B) separates the oral cavity OC from the nasal cavity NC. The nasal concha C (soft tissue which defines, in part, the nasal sinus – not shown) resides in the nasal cavity NC.

10 The soft palate SP (a muscle activated soft tissue not supported by bone) depends in cantilevered manner at a leading end LE from the hard palate HP and terminates at a trailing end TE. Below the soft palate SP, the pharyngeal wall PW defines the throat passage TP. A nasal passage NP connects the nasal cavity NC to the pharyngeal wall PW. Below an epiglottis EP, the throat passage TP divides into a trachea TR for passing air to the lungs and an esophagus ES for passing food and drink to the stomach.

15 The soft palate SP is operated by muscles (not separately shown and labeled) to lift the soft palate SP to urge the trailing edge TE against the rear area of the pharyngeal wall PW. This seals the nasal cavity NC from the oral cavity OC during swallowing. The epiglottis EP closes the trachea TR during swallowing and drinking and opens for breathing.

20 For purposes of this disclosure, the nasal cavity NC, oral cavity OC and throat passage TP are collectively referred to as the naso-pharyngeal area of the patient (defining, in part, the pharyngeal airway PA in Figs. 5 and 7) with the area including the various body surfaces which cooperate to define the nasal cavity NC, oral cavity OC and throat passage TP. These body surfaces include outer surfaces of the nasal concha C, the upper and lower surfaces of the soft palate SP and outer surfaces of the pharyngeal wall PW. Outer surfaces means surfaces exposed to air. Both the upper and lower surfaces of the soft palate SP are outer surfaces.

Snoring can result from vibration of any one of a number of surfaces or structures of the naso-pharyngeal area. Most commonly, snoring is attributable to vibration of the soft palate SP. However, vibratory action of the nasal concha C and the pharyngeal wall PW can also contribute to snoring sounds. It is not uncommon for 5 vibratory action from more than one region of the naso-pharyngeal area to contribute to snoring sounds. Sleep apnea can result from partial or full collapse of the naso-pharyngeal wall during sleep.

Fig. 5 shows a schematic representation of a cross-section of a throat with the pharyngeal airway PA defined by the pharyngeal wall PW and the tongue T. The 10 anterior-posterior axis is labeled A-P to assist in discerning the orientation. The pharyngeal wall PW is shown as including the left lateral pharyngeal wall LLPW, right lateral pharyngeal wall RLPW and posterior pharyngeal wall PPW.

B. Disclosure of Commonly Assigned Applications

15 In addition to disclosing the teachings of U.S. Pat. No. 6,250,307 and the teachings of selected embodiments of PCT International Publication Nos. WO 01/19301 (both incorporated herein by reference), commonly assigned and co-pending PCT International Publication No. WO 02/13738 (incorporated by reference) describes techniques for stiffening tissue of the pharyngeal airway with a bolus of particulate matter. Figs. 2 and 3 show are taken from International Publication No. WO 02/13738 and show an implant 10 as a bolus of particulate matter. An example of such particulate matter would be micro-beads. An example of such is taught in U.S. Pat. Nos. 5,792,478 and 5,421,406. These patents teach carbon-coated metallic or ceramic particles having cross-sectional dimensions of between 100 and 1,000 microns. The particles are carried 20 in a fluid or gel. These patents state that upon insertion into body tissue, the particles do not migrate significantly and, apparently due to fibrotic response, the tissue into which the particles are injected stiffens.

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The particles of U.S. Pat. Nos. 5,792,478 and 5,421,406 are one example of particles for stiffening injection. Such particles can also include ceramic particles or 30 pure carbon or other bio-compatible particles. The particles can be carried in a liquid or gel medium. The particles can have multi-modal particle size distributions (i.e., a mix

of two or more sizes of particles with the smaller particles filling interstitial spaces between larger particles).

The bolus 10 of particles can be applied by a needle to inject the bolus 10 into the soft palate SP. The bolus can be the same volume as the volume of the implants 20 of Figs. 8 and 9 of U.S. Pat. No. 6,250,307. With reference to Fig. 3, a multiple of bolus injections can be made in the soft palate resulting in deposition of generally spherical deposits 10' of particles. Alternatively, an injecting needle can be withdrawn while simultaneously ejecting particles for the bolus 10 (Fig. 2) to be deposited in a line similar in dimensions to the implants 20 of Figs. 8 and 9 of U.S. Pat. No. 6,250,307.

10 The foregoing emphasizes the use of implants to stiffen the soft palate SP. Implants 10 can be placed in any of the tissue of the naso-pharyngeal area (e.g., the concha C, soft palate SP or pharyngeal wall PW) to treat snoring. Also, such a treatment can stiffen the tissue of the throat and treat sleep apnea resulting from airway collapse by stiffening the airway.

15 While a needle deposition of a bolus of particles may be preferred, the bolus can be applied in other manners. Fig. 4 illustrates deposition of particulates through a patch 200 having a volume 202 containing such micro-beads 204.

One side 200a of the patch 200 contains an array of micro-needles 206 communicating with the volume 202. The needles 206 may be small diameter, shallow penetration needles to minimize pain and blood. Examples of shallow, small diameter needles are shown in U.S. Pat. No. 5,582,184 to Erickson et al. Placing the surface 200a against the tissue (e.g., the pharyngeal wall PW as shown in Fig. 4), the needles 206 penetrate the outer surface of the tissue PW. The patch 200 can then be compressed (by finger pressure, roller or the like) to eject the beads 204 from the volume 200 through the plurality of needles 206. The patch 200 can be provided with interior dividing walls (not shown) so that some of the volume of beads 204 is ejected through each needle 206. The side 200a acts as a stop surface to ensure control over the penetration depth of the needles 206 to reduce risk of undesired puncture of underlying structures.

30 Stiffening of the naso-pharyngeal tissue provides structure to reduce vibration and snoring. Such structure reduces airway collapse as a treatment for sleep apnea.

Commonly assigned and co-pending U.S. Pat. Application Ser. No. 10/066,967 filed February 4, 2002 teaches stiffening of the pharyngeal wall with other stiffening techniques including pre-compressing the wall and stiffening the wall in a compressed state.

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C. Transverse Stiffening Implant

Figs. 6 – 7 illustrate the device of the present invention in a pharyngeal wall. As will be further discussed, the implant can be placed at any location along the length of the airway to stiffen the posterior and lateral walls of the pharyngeal wall. While only 10 one implant is shown in Figs. 6 – 7, multiple implants 100 can be placed in the pharyngeal wall as shown in Figure 8.

The device is an implant 110 of biocompatible material selected to induce a fibrotic tissue response. A preferred device is a flexible braid 110 of polyester fibers. In a preferred design, the implant 110 has a diameter of about 2 mm. A length 15 of 10 mm to 20 mm (depending on patient) can cover about 180 degrees surrounding the pharyngeal airway PA. In one embodiment, the implant is elastic so as to be biased toward an at rest position following bending the implant along its longitudinal axis.

The braided implant 110 is preferably formed of a plurality of fibers of similar material (i.e., all polyester fibers). The fibers are bonded together near the ends 112 of 20 the braid to prevent excessive fraying.

The braid 110 may be a simple braid (such as that shown in International Publication No. WO 01/19301 A1 (PCT/US00/40830)) or may be any other braid geometry such as so-called three dimensional braids as described in Du et al., “Geometric Modeling of 3-D Braided Preforms For Composites”, Proceedings of 5th 25 Textile Structural Composites Symposium, Drexel University, Philadelphia, PA (1991) and Ko et al., “Braiding”, Engineering Materials Handbook, Vol. 1, Composites, Reinhart, T.J. Editor, ASM International, Metal Park, OH pp. 519 – 528 (1988). An example of such a more complex braid is shown in commonly assigned and co-pending U.S. patent application Ser. No. 09/814,471 filed March 21, 2001 and filed 30 internationally as PCT/US02/07966 on March 14, 2002, that is hereby incorporated by reference in its entirety.

According to the '471 application, such a 3-D braid could be a 1X1 3-D braid formed on a 24-carrier machine with a braiding angle of 45 degrees and a braid diameter of about 2 mm. The braid is formed of 24 braider yarns of 70 denier yarns with 2 denier/fil (air-textured yarns may be substituted as discussed below). The braid

5 can be formed with 8 axial polyester yarns of 400 denier (7.9 – 8 mil) monofilament. While a core-less braid is preferred, cores can be added for stiffness. Such cores may be 10 monofilaments of polyester at 400 denier per filament. It will be appreciated that 3-D braid construction as described form no part of this invention per se and may be formed as described in the afore-mentioned articles.

10 In a preferred embodiment of a braid as disclosed in the '471 application, the fibers are heat bonded together (e.g., by application of ultrasonic energy) at two locations spaced 1 mm from the ends of the braid. The ends then be frayed for the individual fibers to splay freely from the bonded locations at the ends. The bonded locations insure the fraying does not migrate throughout the entire length of the braid.

15 As an alternative to fraying, the ends may be left braided but unbonded. The ends are then free to fray with the bonded locations insuring any such fraying does not migrate.

With the structure of a braid as disclosed in the '471 application and as described above, the frayed ends present a softened tip to tissue as compared to unfrayed ends of an otherwise identical braid. The softened frayed ends reduce likelihood

20 of migration of the braid in the tissue of the soft palate.

The fibers of the braid may be air textured yarns so that the braid presents a plurality of fiber loops along its length. These loops present increased volume of interstitial space in the braid for increased tissue growth to further reduce the probability of migration of the braid in tissue. Not all of the fibers need be air-textured

25 yarns. For example, 50% (or some different percentage) of the fibers can be untextured (or straight) polyester fibers (or silk or other material) and the remaining 50% can be air-textured polyester fibers (or silk or other material). This ratio can be changed at a braid designer's convenience. Also, as an alternative embodiment, in the braid, air-textured fibers can be placed predominantly in the interior of the braid for a less stiff

30 braid and more interior interstitial space for tissue in-growth.

The implant 110 is placed in the pharyngeal wall PW with the longitudinal axis of the braid 110 bending in the wall PW and extending transverse to the anterior-posterior axis A-P of the airway PA and also transverse relative to a longitudinal axis of the airway PA. The device 110 thus positioned wraps at least partially around the
5 airway PW to reside in the posterior and lateral pharyngeal walls PPW, RLPW, LLPW.

Preferably, the implant is placed in or beneath a mucosal layer. The device can have a radiopaque marker 111 to assist in visualization of the implant during and after placement. With such marker, the device's position and influence on swallowing can be assessed, the device's effect on maintaining the airway patent at various inspiratory
10 pressures can be visualized. The marker and implant should be magnetic resonance imaging (MRI) compatible.

The location of pharyngeal collapse in a patient is identified so that the device can be targeted for the most appropriate location in the pharynx. The majority of airway collapse is believed to occur in the retropalatal region because the airway
15 narrowing has been shown to be greatest in the retropalatal region. Schwab RJ, *Upper Airway Imaging*. Clinics in Chest Medicine 1998; 19(1):33-54. This region is defined as the region from the level of the hard palate to the caudal tip of the soft palate (uvula). However, studies have also shown that collapse also occurs in the retroglossal region, defined from the caudal margin of the soft palate to the base of the epiglottis. See, e.g.,
20 Hudgel DW, *Mechanisms of Obstructive Sleep Apnea*. Chest 1992; 101:541-49.

A body of literature indicates that the lateral pharyngeal walls, rather than the anterior-posterior walls of the pharynx, are the structures which mediate the changes in upper airway size in OSA. Schwab RJ, *Upper Airway Imaging*. Clinics in Chest Medicine 1998; 19(1):33-54; Trudo FJ et al., *State-related Changes in Upper Airway Caliber and Surrounding Soft-Tissue Structures in Normal Subjects*. Am J Respir Crit Care Med 1998; 158:1259-1270; Schwab RJ, *Functional Properties of the Pharyngeal Airway*. Sleep 1996; 19(10):S170-S174; Schwab RJ et al., *Upper Airway and Soft Tissue Anatomy in Normal Subjects and Patients with Sleep-Disordered Breathing*. Am J Respir Crit Care Med 1995; 152:1673-1689. Müller's maneuver with fiberoptic
25 nasopharyngoscopy and cephalometric measurement are presently the key diagnostic tools used to identify the location of airway narrowing causing an apneic episode. See,
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Fairbanks DNF, Fujita S, Snoring and Obstructive Sleep Apnea. Raven Press Ltd., New York, 1994, p. 92. Fiberoptic endoscopy with or without the Müller maneuver has been shown to be able to effectively define the pattern and location of pharyngeal collapse in children. Fairbanks, *supra*, Ref. No. 42, p. 29. However, contrasting literature claims 5 that the Müller maneuver has not been demonstrated to accurately simulate an apneic event during sleep (Schwab RJ, *Upper Airway Imaging*. Clinics in Chest Medicine 1998; 19(1):33-54) and proposes that that a combination of MR imaging and nasopharyngoscopy with a Müller maneuver prior to UPPP may improve outcome after surgery. For example, if the nasopharyngoscopy with Müller maneuver demonstrates 10 retroglossal collapse and the MR imaging demonstrates primarily retroglossal narrowing, surgery directed at advancing the tongue such as geniohyoid advancement or maxillomandibular advancement should be considered rather than UPPP.

Posterior pharyngeal wall movement is observed in swallowing. Jones B, Donner MW, Normal and Abnormal Swallowing. Springer-Verlag, New York, 1991.
15 Schwab has demonstrated that thickening of the lateral pharyngeal muscular walls is the major factor that leads to narrowing of the apneic retropalatal airway. Schwab RJ et al., *Upper Airway and Soft Tissue Anatomy in Normal Subjects and Patients with Sleep-Disordered Breathing*. Am J Respir Crit Care Med 1995; 152:1673-1689.

20 The basis for the increased thickness of the lateral pharyngeal walls in apneics is unknown. Experiments utilizing HUPSEC (Hydrogen Ultrathin Phase-Encoded Spectroscopy) in conjunction with MR imaging indicate that the increased thickness of lateral pharyngeal wall in patients with sleep apnea is not secondary to increased fat infiltration or edema.

Another plausible explanation for the thickening of the lateral walls is that 25 weight gain results in increased muscle mass and an increase in the size of the lateral pharyngeal walls as well as the tongue and soft palate. Therefore, obesity may predispose to sleep apnea by theoretically increasing the size of the upper airway soft-tissue structures rather than by the direct deposition of fat in the parapharyngeal fat pads or by compressing the lateral airway walls by these fat pads. Studies have shown that 30 the parapharyngeal fat pads do not compress the lateral airway walls in apneics.

Schwab RJ et al., *Upper Airway and Soft Tissue Anatomy in Normal Subjects and*

Patients with Sleep-Disordered Breathing. Am J Respir Crit Care Med 1995; 152:1673-1689.

- Apneics have thicker lateral pharyngeal walls when they are awake and these structures become even thicker during sleep. Schwab RJ, *Functional Properties of the Pharyngeal Airway.* Sleep 1996; 19(10):S170-S174. Studies have demonstrated a decrease in the electromyographic activity of the muscles of the lateral pharyngeal walls during sleep as well as the tensor palatini muscle of the soft palate. Trudo FJ et al., *State-related Changes in Upper Airway Caliber and Surrounding Soft-Tissue Structures in Normal Subjects.* Am J Respir Crit Care Med 1998; 158:1259-1270; Wheatley et al, *Influence of Sleep on Response to Negative Airway Pressure of Tensor Palatini Muscle and Retropalatal Airway.* J Appl Physiol 1993; 75(5):2117-2124. The decrease in EMG activity may cause relaxation of these structures, contributing to the lateral wall thickening and airway narrowing. However, thickening of the lateral walls during sleep may be associated with state-related changes (asleep vs. awake) in the conformation of the soft palate and tongue.

A complex three-dimensional biomechanical interaction may exist between the tongue, soft palate and lateral pharyngeal walls. The walls are a complex structure made up of a number of muscles including the hypoglossus, styloglossus, stylohyoid, stylopharyngeus, palatoglossus, palatopharyngeus, the pharyngeal constrictors, lymphoid tissue (palatine tonsils), and pharyngeal mucosa. The biomechanical relationships between these muscles and the way they interact with the soft palate and tongue are not well understood, nor is it understood how the lateral pharyngeal wall moves during respiration or during apnea to modulate airway size.

Whether the airway actually collapses laterally or A-P is not discussed in the literature. MR anatomical studies demonstrate posterior movement and thickening of the soft palate with sleep as well as lateral pharyngeal wall thickening. Trudo FJ et al., *State-related Changes in Upper Airway Caliber and Surrounding Soft-Tissue Structures in Normal Subjects.* Am J Respir Crit Care Med 1998; 158:1259-1270. Other studies have shown that increasing increment levels of effective CPAP therapy results in increased airway volume and area within the retropalatal and retroglossal regions, greater lateral than A-P airway dimensional changes, a decrease in the lateral

pharyngeal wall thickness, and an increase in the distance between the lateral pharyngeal fat pads. Schwab RJ, *Functional Properties of the Pharyngeal Airway*. Sleep 1996; 19(10):S170-S174. Furthermore, effective mandible repositioning devices have been shown to thin the lateral pharyngeal walls; it is theorized that these devices 5 may put traction on the lateral walls, resulting in the thinning of the walls. Subsequently, designing a device that affects the lateral pharyngeal walls seems appropriate in effectively addressing OSA.

- An effective device preferably establishes a pressure differential of 8 cm H₂O between nasal pressure and pharyngeal critical pressure (P_{crit}) to eliminate obstructive 10 sleep apnea and snoring. The critical pressure is defined as the pressure that is equal to the pressures exerted on the airway by the surrounding tissue which is equal to the pressures tending to collapse the airway See, Gold AR, Schwartz AR, *The Pharyngeal Critical Pressure*. Chest 1996; 110:1077-88. It is determined by finding the level of nasal pressure below which maximal inspiratory airflow ceases (airway collapse). 15 Schwarz AR et al., *Effect of Uvulopalatopharyngoplasty on Upper Airway Collapsibility in Obstructive Sleep Apnea*. Am Rev Respir Dis 1992; 145(3): 527-532. A higher P_{crit} equates to an increase in collapsibility. The P_{crit} for normal people is < - 8 cm H₂O. For snorers the P_{crit} is approximately - 4 cm H₂O, and for those with OSA (obstructive sleep apnea), the P_{crit} is approximately 0 cm H₂O. See, Gold AR, supra. 20 The implant should be able to withstand a pressure of 4.4 cm H₂O to prevent collapse. This was the upper airway suction closing pressure found in OSA patient studies by Issa FQ, Sullivan CE, *Upper Airway Closing Pressures in Obstructive Sleep Apnea*. J Appl Physiol 1984; 57(2):520-527. Studies have shown that by sleeping with one's mouth open, a 4 cm H₂O increase in P_{crit} can occur. See, Gold AR, Schwartz AR, 25 *The Pharyngeal Critical Pressure*. Chest 1996; 110:1077-88. Effective UPPP surgery has demonstrated a reduction in P_{crit} from -0.8 to -7.3 cm H₂O. Schwarz AR et al., *Effect of Uvulopalatopharyngoplasty on Upper Airway Collapsibility in Obstructive Sleep Apnea*. Am Rev Respir Dis 1992; 145(3): 527-532.

- Normal individuals maintain a positive pharyngeal transmural pressure of 30 approximately 0 to 10 mm Hg (13.6 cm H₂O) during sleep. In contrast, upper airway obstruction in apneics is associated with development of negative transmural pressure

during sleep. Clinically significant levels of obstructive sleep apnea can be produced when a nasal pressure of -10 cm H₂O is applied to normal subjects during sleep. These findings suggest that a disturbance in reflex responses is not necessary for the development of recurrent obstructive apneas (as some literature suggests). Rather,

5 structural or neuromuscular factors that decrease the pharyngeal transmural pressure will predispose to this disorder. King ED, O'Donnell CP, Smith PL, Schwartz AR, *A Model of Obstructive Sleep Apnea in Normal Humans*. Am J Respir Crit Care Med 2000; 161:1979-1984.

The dynamic biomechanical relationships between the upper airway muscles

10 that allow physiologic functions like vocalization, swallowing, and respiration to occur is complex. Schwab RJ, *Upper Airway Imaging*. Clinics in Chest Medicine 1998; 19(1):33-54. In the pharyngeal phase of swallowing, more than two dozen muscles are required to function together to effectively transport a bolus from the mouth to the esophagus. The posterior pharyngeal wall movement has been studied by Palmer et al.,

15 there is considerable mobility normally present in the posterior pharyngeal wall (retropalatal and retroglossal). Palmer JB et al. *Motions of the Posterior Pharyngeal Wall in Swallowing*. Laryngoscope 1999; 98:414-417.

The implant is sized to be implantable in a typical apneic based upon the following anatomical measurements (Schwab RJ et al., *Upper Airway and Soft Tissue Anatomy in Normal Subjects and Patients with Sleep-Disordered Breathing*. Am J Respir Crit Care Med 1995; 152:1673-1689):

- a. thickness of lateral pharyngeal wall: (approx 30 mm)
- b. anterior-posterior diameter of airway: (4.7 +/- 2.5 mm)
- c. lateral diameter of airway (6.7 +/- 4.5 mm) at the level of the minimum airway area in apneics.

The foregoing describes numerous embodiments of a method and apparatus to treat a pharyngeal wall. Having described the invention, alternatives and embodiments may occur to one of skill in the art. For example, while placing one or more implants 110 in the pharyngeal wall partially extending around the circumference of the pharyngeal, is a presently preferred embodiment, other alternatives exist. For example, implants 110' can be placed in pharyngeal wall parallel to the axis of the pharyngeal

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airway and spaced around the circumference of the pharyngeal wall as shown in Figures 9 and 10. Also alternatively, a sheet of polyester material 110" (or other fibrosis-inducing material) can be placed in the pharyngeal wall at least partially surrounding the axis as shown in Figure 11. In all embodiments, any stiffness of the material and 5 any resulting fibrosis add structural integrity to the pharyngeal wall. It is intended that such modifications and equivalents shall be included within the scope of the following claims.